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Treatment of Pulmonary Consumption ××

THE TREATMENT OF PULMONARY CONSUMPTION, WITH A REPORT OF FORTY CASES.

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This paper is devoted to a consideration of the therapeutics of pulmonary consumption, but the therapy of any disease is meaningless unless the underlying pathological conditions are correctly interpreted, and we shall, therefore, at the very outset, endeavor to outline, in as brief a manner as possible, the recognized principles of the pathology of this disease, and then follow with a discussion of those measures which experience has proven to have the power of controlling and of dissipating it.

Now, pulmonary consumption is generally an extension of chronic bronchitis into the alveoli, or is the product of acute catarrhal pneumonia. In either case, it is a catarrhal affection of the alveolar epithelium. The pulmonary capillaries become engorged, and the epithelial cells multiply and accumulate, and clog up the alveoli with their products. The filling up of the alveoli with these catarrhal aggregates produces small bodies which partake of the shape and size of the former. In this way one alveolus fills up after another, until a whole group or a cluster of them is involved, giving rise to roundish nodular bodies, which are so frequently mistaken for tubercles. They are not tubercles at all, in the technical meaning of that term, but are mere *accumulated inflammatory or catarrhal products*. This train of pathological changes is due to a disturbance of the relationship existing between the production and expulsion of epithelial products. If such a relationship were preserved, or could by any means be restored, it is evident that the disease would at once be called into abeyance. Thus far, the process is chiefly limited to the alveolar walls, their epithelium and their blood-vessels; but the continued accumulation of catarrhal products exerts a decided pressure on the surrounding pulmonary and bronchial capillaries, and the blood supply and nourishment are gradually diminished and finally cut off from these infiltrated areas, which, in due course of

time, become isolated and circumscribed foreign masses, prone to undergo a slow process of cheesy degeneration, and leave behind cavities, large or small, according to the amount of tissue destruction.

It is during this stage of excavation that the true tubercle is brought to life. A specific poison is derived from the caseating catarrhal nodules, which is chiefly absorbed by the lymphatics. These vessels transport this specific element along their ascending courses as they arise in the alveolar wall, and twine around the walls of the blood-vessels and the bronchioles, and it is here, in the beginning, and in the channels of the lymphatics, that this poison incites new nodular growths, which are *genuine tubercles*, but differ from the yellow, or catarrhal nodules, already considered, both in genesis and in structure. They are evolved from interstitial connective or lymphatic tissue, and are growths, or a hyperplasia, and not mere epithelial or catarrhal aggregations like the so-called yellow tubercles.

It must be distinctly understood, however, that pulmonary consumption is such an intensely sectional disease that what has been said so far in regard to the catarrhal origin of the disease pertains almost exclusively to the apices of the respiratory organs. And it may, therefore, be laid down as a safe rule in the diagnosis and prognosis of chest diseases, that, in by far the great majority of cases, partial or complete consolidation of an apex denotes the first stage of pulmonary consumption. While it is not absolutely true that every case of apical infiltration terminates in pulmonary cavitation, it is enough for the practitioner to know that it is the beginning of a process which, if not checked, certainly tends to end there.

It is very evident, therefore, that the focus of disturbance in pulmonary consumption is an infiltration of the vesicular structure of the lung, and a consequent consolidation, and that the one great desideratum in its treatment is as to the best method of removing this; or, in other words, how is resolution of the affected part to be accomplished? In croupous pneumonia, the intra-alveolar infiltration, which consists principally of a fibrinous infiltration, is quickly removed through a process of fatty degeneration; but in catarrhal pneumonia the infiltration does not consist of fibrinous exudation, but, as we have seen, of an accumulation of epithelial products, and, instead of tending toward fatty degeneration, it undergoes a

process of caseation. The difference between fatty and cheesy degeneration is the line which divides the fatal nature of the latter from the non-fatal nature of the former disease; and if the therapeutic art can devise a method by which the catarrhal infiltration can be resolved into a fatty or into a calcareous instead of into a caseous degeneration, the problem of the treatment of pulmonary consumption will be forever solved.

The great obstacle to the successful treatment of pulmonary consumption, however, is the inability to introduce agents which reach and act on the focus of the disease, viz., on the seat of infiltration. Efforts have been repeatedly made to apply medication locally through the air passages by inhalation, as well as through the blood, either by hypodermatic injection or by gaseous enemata, according to the now famous method of Dr. Bergeon. But we have already seen that the most pronounced seat of infiltration is entirely beyond the reach of bronchi or blood-vessels, hence it cannot be expected that any of these methods have the power to antagonize the citadel of the disease, however capable they may prove themselves in removing some of its concomitants.

We have here then, in most instances, a condition which, to all intents and purposes, is a deposit of inflammatory products differing, in principle, in no wise from a similar deposit in any other part of the body, and the dictates of common sense point out that that which is useful in the one condition is also useful in the other. Every physician can testify to the inestimable value of counter-irritation, and of passive motion in producing resorption of chronic inflammatory deposits in joints, and in the external surfaces generally; and in consonance with this view we have, for a number of years, applied hot flaxseed-meal poultices, as well as friction to the chest, in such affections, and, we believe, with the most gratifying results. There is reason for believing that these external applications have the power of increasing the circulatory, the lymphatic, and the cellular activity of the affected part, and, in this manner, gradually remove the most pronounced pulmonary infiltration.

The manner of making these external applications will be briefly described here. First then as to the poultice: This is made by boiling the ground flaxseed meal in a sufficient quantity of water to make a thick paste. Spread it quickly in a layer an inch thick between two pieces of flannel cut in the shape of a jacket, which is

well fitted to the top, front, and back of apex, as well as to the anterior and posterior aspects of the affected lung. The edges are sewed or tied together, then it is adjusted to the chest, and the outside well covered with oiled-silk, or some thick woolen material, in order to prevent a too rapid radiation of heat. It must be changed every two or three hours, or sooner if it becomes cooler than the body. This process is to be continued from morning until night, when the poultice is taken off, and the chest is well wrapped with woolen flannel for the night, in order to avoid a sudden disturbance of the body temperature during the sleeping hours. The next morning the same programme is begun and continued in the same way for at least three weeks, and so much longer as is necessary. It is much more expedient to poultice during the day only, and allow the patient to rest and be free from the poultice during the night. In fact, this is very important, for if you insist on poulticing during the night also, you are very apt to worry and discourage the patient, and get him to refuse the application altogether.

In a course of five or eight days poulticing, or perhaps sooner, the change in the physical signs will become most marked. Where previously there was heard nothing but a rough or bronchial respiration, sub-crepitation, perhaps crepitation, and mucous râles show themselves, leading one to suspect that the disease is advancing instead of receding. In the course of a short time time, however, the whole part will clear up and become dry. Associated with these changes in the physical signs, is an improvement in the patient's general condition. He experiences more freedom in his respiratory movements, the cough becomes easier, the expectoration looser, the appetite begins to improve, and he coughs less at night and sleeps better.

In addition to the application of moist heat, we also employ local or general massage once or twice a day. This has the happy effect of counteracting the constitutional lethargy present in phthisis, as well as arousing the local cell activity in the pulmonary organs. Dr. T. Henry Green, in his lectures on the "Pathology of Pulmonary Consumption," delivered in the Brompton Hospital for Consumption, in 1878, speaking of external treatment in this disease, says (p. 102):

"That in these last named cases (cases where much intra-alveolar matter exists which is capable of removal) attempts to stimulate the circulation in the diseased lungs by douches and friction, and other similar means, such as have been recently advocated by a Dr. A. von Sokolowski,

would appear to me to be more in accordance with the teachings of pathology."

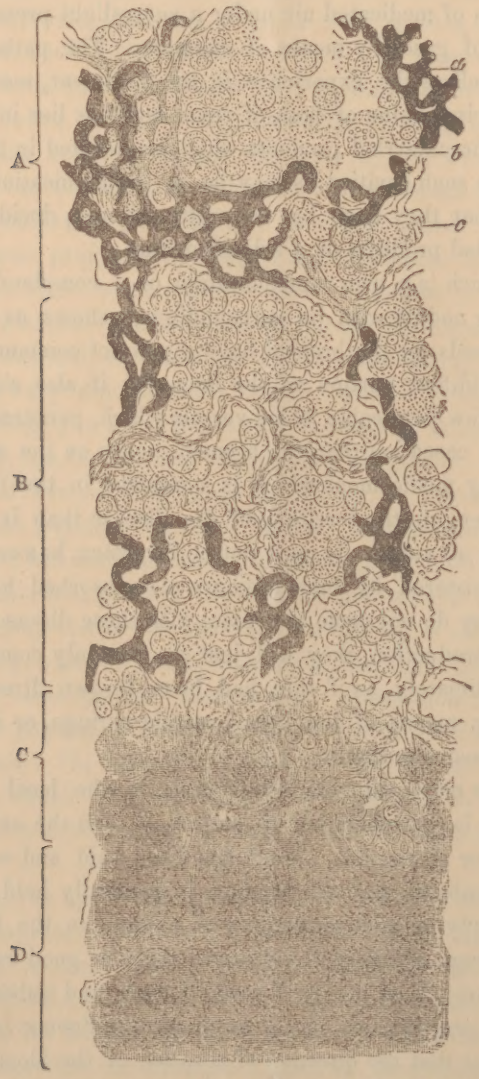
Next in importance in the local treatment of this disease, comes the inhalation of medicated air under a very slight pressure, and the introduction of gases by means of enemata. The pathology of the disease, as well as the experience in its treatment, teach, however, that the principal value of both of these methods lies in their power to disinfect the catarrhal products which are located in the bronchial tubes, and in such cavities as may be in communication with these tubes, and that they have but very little, if any, decided influence on the catarrhal products of a solidified lung.

Fig. 1, which is a true representation of a completely infiltrated and caseating nodule with its surroundings,* shows us that neither the blood-vessels nor the bronchi have any direct communication with the truly solidified portion of the lung, yet it also shows us that there are a few permeable blood-vessels which penetrate nearer to the centre of caseation than the bronchi, hence on the surface it apt pears that any medicament which is contained in the blood will be carried further into the territory of the disease than it will be conveyed by the air tubes. It must not be forgotten, however, that substances administered by inhalation are also absorbed by the blood, and while they do not come in contact with these diseased centres on their first introduction, they will just as certainly come in contact with them ultimately, as if they had been thrown directly into the circulation by means of the hypodermatic syringe, or indirectly by means of gaseous enemata.

One of the most valuable acquisitions to the local treatment of chest disease is the inhalation of compressed and the exhalation into rarefied air, or *vice versâ*. Much has been said and written in regard to this subject, and the opinion is generally held that it is a means of supplying a larger amount of oxygen to the blood than is obtained through ordinary respiration; there is good reason for believing, however, that nearly the whole sum and substance of the value of this practice lies in its mechanical influence on the lungs. If it were true that the question of aëration of the blood was simply one of diffusion of air in the lungs, and not one of a chemical affinity between the hæmoglobin and the oxygen, undoubtedly more oxygen

* On Catarrhal Pneumonia and Tubercle in the Human Lung, by D. J. Hamilton, M. B., F. R. C. S. Ed. Practitioner, vol. xxiv., page 258.

could be utilized by the lungs under this procedure. At the same time it cannot be denied that by a more complete expansion of the



Catarrhal pneumonia, second stage, showing a nodule magnified 350 diams. A, B, C, and D, represent different areas in the nodule from the periphery towards the centre. *a*, injected capillaries of alveolar wall; *b*, catarrhal cells in alveolar cavities; *c*, an alveolar wall.

lungs under such circumstances, a larger quantity of hæmoglobin will be exposed, and will combine with or absorb more oxygen than under

ordinary atmospheric conditions, yet from all the experimental data which we have on this point, it is very probable that the increased absorption is very slight.

Any one who has given the method a faithful trial has found abundant evidence of its usefulness in nearly all forms of chest diseases, and especially so in the treatment of the disease under consideration. It gives complete ventilation to, and increases the capacity of, the chest; it relieves the dyspnœa, and acts as a powerful tonic to the whole body. It should be employed with great care, and not be used during a high temperature, nor in an active inflammatory condition of the lungs, except under the very lowest pressure. Indeed, it seems that we have obtained the very best results from it after the activity of the disease had receded, and when convalescence was well under way.

Another important method in the local treatment of this affection is the application of the constant electric current to the chest, according to the recommendation of Dr. Bastings, of Brussels. This procedure consists in chiefly employing the ascending current, and it has special value in those cases in which there is associated with the internal trouble immobility of the thorax, myalgia, or intercostal neuralgia.

In connection with the local treatment we must not lose sight of the marked benefit which may be derived from internal treatment, especially in so far as the question of supplying force and matter to the body is concerned. Pulmonary consumption is preëminently a wasting disease, not necessarily of the lungs but of the whole frame, and any food or medicine which supplies this constant waste, or facilitates digestion and assimilation, is very strongly indicated, and for this reason nutritious foods, cod-liver oil, hypophosphite preparations, etc., are of such great value, if used with discretion. So far as the force-producing value of foods is concerned, there can be no question that cod-liver oil and the fatty and saccharine foods rank very high, but very frequently we find an aversion to the first, and ermentation following the ingestion of the last two; and hence they must be in a great measure avoided. The non-nitrogenous foods which are least likely to undergo fermentation are cream, butter, and alcohol, and the dietic value of these articles increases in proportion as it becomes necessary to exclude the other foods of this class from the dietry list. The best nitrogenous foods are beef, mutton, eggs, oyster, milk, bread, peas, beans, etc. Animal food often

renders the best service when it is chopped into a pulp and consumed when raw. In cases of complete anorexia forced feeding, either by the stomach or by the rectum, must be resorted to, and the food administered regularly three or four times a day, with the addition of a few drops of muriatic acid and a little pepsin. Milk alone does surprisingly well sometimes in this form of feeding.

We have the records of forty cases which have been treated in accordance with the plan here advocated, and we believe that the practical results which it achieved warrants us in recommending it to our fellow-practitioners. The first twenty of these cases were reported by us in a contribution to the *Medical News* of October 10, 1885, and we take pleasure in stating that the prognosis which we then gave in regard to these cases has been fully confirmed by subsequent observation. Of the entire forty cases thirty-one recovered. The sixteen of the first twenty cases who recovered have been under occasional observation during a period of from two to nine years, and so far as we know, are all well at the present time. The following gives the condensed histories of the forty cases thus treated:

Case I. Dates from May 1, 1879, and was in the first stage. Cough, and profuse expectoration streaked with blood for nine months. Hæmoptysis, chills, night-sweats, loss of flesh, and poor appetite. Diminished percussion resonance, and crepitation over left apex. She was treated for a month. Afterwards married, became the mother of a family, and is well at present.

Case II. First seen in March, 1879. Suffered from loss of sleep, constant cough, profuse expectoration, night-sweats, poor appetite, coated tongue, wasting of flesh. Family history of phthisis. Dulness and crepitation in upper two-thirds of left lung. Made a complete recovery.

Case III. Began treatment in November, 1880. Cough, blood-streaked expectoration, stitches in left apex, night sweats, wasting and poor appetite. Lost a brother from phthisis. Diminished percussion resonance. Crepitation and wavy respiration in left apex. Made a good recovery.

Case IV. Dates from July, 1883. Cough, chills, very thin, poor appetite, no hæmoptysis. Weight, 100½ pounds. Phthisis in family. Dulness and crepitation in left apex. Gained eight pounds and a half in two months, and is well to-day.

Case V. A boy four and a half years old. First seen July,

1883. Cavity in left apex, and more or less consolidation to base of same lung. Made a slow but good recovery.

Case VI. Was in third stage when she came under treatment in August, 1883. Copious hæmoptysis, lost flesh rapidly, much cough and expectoration, and a poor appetite. No hereditary history. Depression and tympanitic sound over subclavicular region, right side, mucous râles from here to base of lung. Coarse mucous râles distributed over posterior base and interscapular region of left lung. In one month she gained nine and a half pounds. She is well now.

Case VII. Began treatment in September, 1883. Had profuse hæmoptysis, great emaciation, poor appetite, and a family history of the disease. He was in the third stage, and weighed 133 pounds. Tympanitic sound over left apex, both anterior and posterior, with slight dulness under third rib. Cavernous and mucous sounds over same region, with a border of crepitation at its lower limit. Prolonged expiration in right apex. In the following January the moist râles had entirely disappeared from both lungs, and his weight had increased three and a half pounds. There was, however, no permanent improvement in his appetite, and he finally sank, and died in the spring of 1885.

Case VIII. Came under observation in August, 1883. Had a great deal of cough, profuse expectoration of dark-green color and streaked with blood. No hæmoptysis, night sweats, or chills. He was in third stage, and weighed 128¾ pounds. Left lung, tympanitic sound from apex to nipple, and flatness from here to base. Cavernous sounds from clavicle to nipple, and complete absence of respiration over region of flatness, anteriorly, posteriorly and laterally. Right lung normal. In the following April he weighed 137 pounds. The left lung at this time was permeable and had resumed its respiratory function in a moderate degree, and the cavity became dry. He is still alive, and considering the amount of tissue destruction he is doing very well.

Case IX. First seen in January, 1884. Had cough, hæmoptysis, night sweats, poor appetite, and vomiting. Impaired percussion, resonance and crepitation in right apex. He improved somewhat, but had a relapse, and a cavity formed under the right clavicle. He was a pauper, and frequently suffered for the necessaries of life, and from last accounts he is still alive, yet he will in all probability die.

Case X. Was in the first stage with hereditary tendencies when

treatment was begun in January, 1884. Had cough, copious expectoration and hæmoptysis. Dulness and crepitation in left apex. In a month and half every abnormal physical sign had disappeared.

Case XI. Came under observation in August, 1884. She had frequent hemorrhages, was much emaciated, poor appetite, night sweats, chills, and high fever. Barrel shaped chest, with depression below both clavicles, cavity in left apex, dulness, crepitation and cavernous râles in upper half of left lung. In the course of three weeks she slept better, coughed and expectorated less, appetite much improved, and evening temperature but slightly above the normal. At this time there were no moist or crepitant râles in any part of her chest. The following December all her chest symptoms became aggravated, and she died in the same month.

Case XII. Dates from January, 1885, Constant cough, yellow and blood-streaked expectoration, chills, fever, and night sweats. Dulness in right apex, cavernous râles directly beneath clavicle, and mucous râles to third rib. Bronchial respiration over posterior aspect of same apex. In the course of six weeks, the moist râles had disappeared, and a small cavity showed itself beneath clavicle. Was seen a few months ago, and barring the cavity sounds there were no abnormal chest signs present.

Case XIII. Came under observation in January, 1885. Had a severe cough, yellow expectoration, hæmoptysis, night sweats, losing flesh, and poor appetite. Dulness and increased vocal resonance and fremitus over right apex. Bronchial respiration posteriorly, and mucous râles anteriorly, over same region. In the following March (early part) her chest was free from râles. She was seen a few months ago and examined, and there is every evidence of a complete recovery.

Case XIV. Was seen first in February, 1885. He had a rickety, pigeon-shaped chest, with a deep retraction of the lower end of sternum. Had a number of copious hemorrhages, and lost flesh rapidly. Respiratory motion wavy and very feeble. There was dulness and crepitation in right apex anteriorly and posteriorly. Patient gave a specific history. In April examination showed his chest to be free from all abnormal physical signs except a slightly diminished resonance and a coarse vesicular sound in right apex, and he then resumed his work as a printer. In the following July he had a relapse: hæmoptysis and crepitation re-appeared in the same

lung, which gave way again to the same treatment. Last winter he became worse, pyo-pneumothorax supervened, and in all probability he is dead by this time.

Case XV. Was first seen in May, 1885. Dulness, crepitation, bronchial respiration, and moist râles in lower half of left lung In three weeks the râles had all disappeared, but some impairment of resonance remained. Patient is well and able to work at the present time.

Case XVI. Came under treatment in May, 1885. Dulness, bronchial respiration, and crepitation over right apex. In beginning of June crepitation had entirely disappeared, and bronchial breathing and dulness were much less marked. From last accounts he is well and at work.

Case XVII. Dates from May, 1885. Had a cough, yellow expectoration, hæmoptysis, and the only surviving member of a family of eleven children, all the rest having died of consumption. Dulness in left apex extending to second intercostal space in front, and to spine of scapula behind, with marked crepitation in supra-clavicular region. In the middle of June nearly all trace of the disease had disappeared, and she continues well up to the present time.

Case XVIII. Was placed under treatment June, 1885. Constant cough, worst at night, yellow expectoration, night sweats, chills and fever, losing flesh and no appetite. Dulness in left apex, prolonged expiration posteriorly, and wavy respiration anteriorly in same region. In a month all abnormal physical signs had disappeared, and she remains well up to the present.

Case XIX. Was first treated in May, 1885. He then had cough, blood-streaked expectoration, chills, loss of flesh, and no appetite. Dulness and crepitation in right apex. In the following June very little evidence of abnormal signs existed. He made a good recovery.

Case XX. Presented the following condition in June, 1885. Constant cough, dark greenish expectoration, hæmoptysis, dulness and prolonged expiration over right apex. She improved markedly both in symptoms and physical signs, and a late examination shows that her chest is free from abnormal sounds.

Case XXI. Was first seen June 1st, 1885. Had hæmoptysis, dyspnœa, cough and expectoration. Loss of flesh and progressive weakness, night sweats. Family history of phthisis. Dulness in left apex posteriorly, cog-wheel respiration anteriorly. He improved

at once, and on Sept. 23d his physical signs were normal and he felt very good. He continues well at present.

Case XXII. Came under observation Oct. 14th, 1886. Cough for twenty-five years, one hæmorrhage two years ago. Expectoration profuse and yellow, losing flesh. Weight 129½ pounds. Dulness left apex to second rib as well as to apex of scapula behind. Crepitation in same region ant. and post. mixed with mucous and sibilant râles, roughened respiration distributed over remainder of lung surface. Oct. 26th he had gained one pound, and the moist râles had markedly diminished. The following 2d of November he went home, at which time he had gained another pound and the abnormal physical signs had very nearly all disappeared. Have not seen this patient since, but a month ago he wrote that he has not felt so well in a long time as he is doing now.

Case XXIII. Began treatment 30th of December, 1886. Coughed for six months, copious expectoration. Dulness in left apex, accompanied with crepitation. January 25th, 1887, she felt well, and every trace of disease had disappeared from her lung.

Case XXIV. Came under treatment October 22d, 1885. Cough and expectoration, hæmoptysis four years ago, poor appetite. Strong family history of phthisis. Subcrepitation and tubular respiration in right apex. November 5th, râles and bronchial respiration very much diminished. 17th of November she feels very good, and her chest signs are normal.

Case XXV. Was first seen January 12th, 1885. Coughed, had hæmoptysis. Weight, 126 pounds. Dulness in left lung anteriorly and posteriorly, crepitation extending over apex from nipple in front to apex of scapula behind. Vocal fremitus and resonance increased over same region. Right lung normal. Traces of an old pleurisy in left side. Mitral regurgitation. October 31st of same year the physical signs in his chest were normal except the mitral murmur, and his weight was 134 pounds. The lung affection in this case was secondary. In the following spring he began to suffer with much pain over the cardiac region, his lungs became worse, and he gradually became weaker and died.

Case XXVI. Came under observation September 29th, 1885. Complained of pain in chest, and constant dyspnœa for about five months. Number of respirations 76 per minute. Cough, expectoration yellow and very copious, hæmoptysis, losing flesh, appetite

poor. Family history of lung trouble. No dulness. Large and small mucous râles diffused in abundance over whole chest. November 12th, cough and expectoration very little, respiration 22 per minute. Mucous râles almost entirely gone.

Case XXVII. Was first treated September 29th, 1885. Coughed and expectorated freely. Losing flesh. Dulness in right apex, increase of vocal resonance and fremitus in same region. Crepitation in supra-scapular and supra-clavicular region. October 17th, dulness diminished and crepitation gone. Feels very good. March, 1886, he had gained twenty-five pounds.

Case XXVIII. Began treatment June 1st, 1885. She has a cough, copious expectoration streaked occasionally with blood, night sweats. Normal weight 120 pounds. Weight two months ago 98 pounds, present weight 93 pounds. Family history of phthisis. Dulness in right apex extending to first intercostal space in front, and to spine of scapula. Prolonged expiration and a click toward end of inspiration in same region. Left lung normal. June 26, dulness certainly less, prolonged expiration is improved, and the click has disappeard. Gained five pounds. From last accounts she is very well.

Case XXIX. Was first seen August 9th, 1885. Had a chill the previous September, and coughs ever since. Yellow expectoration, occasionally greenish, no blood. Dulness in right apex. Vesiculo-bronchial respiration in anterior aspect of apex to fourth intercostal space and posteriorly to spine of scapula. Sibilant and a few mucous râles heard over base of same lung. Left lung normal. October 12th, felt much better. Dulness diminished. Prolonged expiration, mucous and sibilant râles have entirely disappeared. May 12th, 1886, exposed herself, and had a fresh attack. From this time on she became weaker, and died the following August.

Case XXX. First seen in October, 1885. Had been losing flesh for about two months, a chill three weeks before which was followed by fever. Blood streaked expectoration, color of expectoration at first was yellow, but changed to green-dark; phthisis in his family. Impaired percussion resonance at base of left lung. Sibilant râles at base of both lungs, the same are also distributed over whole lower third of right lung. Nov. 2d, physical signs normal. Gained in flesh and feels very good. Is doing well at present.

Case XXXI. Came under observation in Nov., 1885. Had

copious hæmoptysis four years before, cough and copious expectoration. Strong family history of phthisis. Dulness in lower half of right lung extending up to 3d rib. Some moist râles distributed over same region. Left lung healthy. This patient was never seen after first examination, but understand he carried out the treatment and is very well now.

Case XXXII. Dates from December, 1885. Cough, deep yellow and profuse expectoration, dyspnœa, and history of phthisis in family. Dulness in left apex. Broncho-vesicular respiration in same apex. Sibilant and mucous râles heard over whole of same lung. By middle of following January she was free from all adventitious sounds in her chest, except a slightly impaired percussion resonance in left apex. She is doing well at the present time.

Case XXXIII. Was first seen March, 1887. Trouble began with a chill, which was followed by a cough and blood streaked expectoration. Dulness in left apex from apex to 3d rib in front, and to spine of scapula behind, crepitation in same region. May 5th all the abnormal physical signs had vanished except the dulness, which however had been markedly diminished. She is doing very well now.

Case XXXIV. Dates from October 18th, 1886. Cough for nearly a year, copious expectoration, loss of flesh and failing strength, appetite poor, night sweats, hæmoptysis. No history of phthisis in family. Chest extremely emaciated. Tympanitic or cavity sound from apex to third intercostal space, dulness from here down. Blowing inspiration and expiration above third rib, pitch of expiration lower than that of inspiration, crepitation over area of dulness below third rib. Right lung normal except prolonged expiration in apex, Nov. 9th. Feels much better, appetite very good, no night sweats. Crepitation has disappeared, other physical signs as before. April 26th, 1887. Feels much better, gained in flesh, and thinks the poultice has helped him very much.

Case XXXV. Came under treatment February, 1886. Had a cough, expectoration and blood-streaked sputum. Dulness at base of left lung, associated with crepitant râles. In a month the râles had disappeared, and the dulness had perceptibly diminished. She is well now.

Case XXXVI. Began to be treated July, 1886. Had some cough and expectoration, no hæmoptysis, poor appetite. Body very

much emaciated. Slightly impaired percussion resonance of left apex. Few mucous râles, with occasional clicks in same apex. Under treatment her left lung became normal again, but she failed to gain in flesh. She became subject to severe attacks of pain radiating from her left ovary over the whole abdomen. Similar attacks of pain diffused themselves over the chest, especially on the right side. The right lung finally became involved, and she gradually became weaker and died last March.

Case XXXVII. Came under treatment October 9th, 1886, with cough, yellow expectoration, hæmoptysis, losing flesh, night sweats, and poor appetite. Impaired percussion resonance in left apex to third rib. Blowing inspiration over anterior aspect of this region, mucous and subcrepitant râles from third rib to base of lung. Right lung normal. In a month she felt very good, and the physical signs had nearly all cleared away. She is now well, and has resumed her occupation.

Case XXXVIII. Was first begun to be treated 24th of March, 1887, when he suffered from constant dyspnœa, coughed, had yellow expectoration, hoarseness, night sweats, and lost flesh since the preceding August. Dulness in right lung from apex to second rib in front, and to spine of scapula behind. Crepitant and sibilant râles associated with bronchial respiration in anterior aspect of apex over area of dulness, while from here down to base of lung mucous râles. Left lung, mucous râles distributed over anterior surface. Thus patient was treated exclusively for two weeks with the gaseous enemata without any perceptible improvement; after which he was poulticed and massaged, and in the course of six weeks' treatment the dulness in right apex had almost entirely disappeared as well as the râles. The whole chest is now nearly dry, and he feels strong enough to resume his work.

Case XXXIX. Was first seen October, 1886, when he coughed and expectorated and had been losing flesh for 6 months. Impaired respiratory movement of thorax. Dulness in right apex extending to third rib in front and to apex of scapula behind. Crepitation, subcrepitation and bronchial respiration over whole area of dulness, anteriorly and posteriorly, and mucous râles extending from here to base of right lung. Crepitation at base of left lung anteriorly. In less than five weeks he gained thirteen pounds, at which time there was decidedly less dulness, and a general disappearance of rales in the chest. He is well and at work now.

Case XL. Aged 58, a cloth-finisher came under treatment in September, 1886, when he had but little cough, not much expectoration, hæmoptysis the same day he was first seen, losing flesh and no appetite. Slight dulness on percussion, left apex blowing expiration and a few moist râles in same region. Strong family tendency to phthisis. Weight 131 pounds. In the course of a short time he improved in every respect; gained 6 pounds in less than two months; went to work again at his old occupation against our advice. March 12th, 1887, he spat up some more blood, and from that time on he sank, and died in a month and a half after the last date.

In conclusion, we beg to state that these forty cases do not embrace the whole number which we treated according to this system of medication, but, with a few exceptions, include all those of which we possess a complete history in each individual case, from the beginning of the treatment up to the time of death or to that of recovery. The considerable time which has elapsed in the majority of these cases since suspension of treatment is an important indication as to the value of the latter. We do not by any means parade these statistics with a view of showing that this is a new means for resurrecting the dead consumptive, nor are we unmindful of the fact that just as long as the professional mind is willing to be blown about by every passing therapeutic breeze, so long will a successful solution of the treatment of this disease remain futile; but we earnestly believe that the system which we advocate here, and which has withstood the test of experience in our hands for nearly half a score of years, offers greater prospect of success than any other, and shows that a great deal more can be, than has been, done for this unfortunate class of patients.